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Electrocardiographic diagnosis of coronary artery disease

Ellet H. Drake

University of Nebraska Medical Center

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THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF
CORONARY ARTERY DISEASE

Ellet H. Drake

SENIOR THESIS PRESENTED TO THE COLLEGE OF MEDICINE

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INTRODUCTION

While the application of electrocardiography to the diagnosis of coronary artery disease is comparatively recent, the modern method of the graphic registration of the heart beat is the result of many years of research and experimentation. As early as 1856, Muller and Kolliker, using the heart of a frog, demonstrated that the contraction of the heart muscle was accompanied by a difference in electrical potential. Waller, working in his experimental laboratory in 1887 (169) demonstrated that these differences in potential could be recorded by means of a capillary electrometer if proper contact could be made between the wires of the electrometer and any two areas of the body which included the heart between them. It was not until 1903, however, that Einthoven of Leyden introduced the string galvanometer which has been slowly developed into the sensitive electrometer of today thus putting at our disposal an accurate means of studying the various heart disorders by graphic methods. (32)

The electrocardiogram is a record of the production of electricity by the heart muscle. This electrical production is one of the functions of the muscle and is intimately connected with the contraction of the fibers. Thus if the muscle is diseased, it will produce a different graphic picture of the changes in electrical potential than will be produced normally. Due to the fact that the graphic record in disease will differ from that obtained from the healthy heart muscle, it is possible to diagnose many types of myocardial disorders through correlation of certain abnormalities

of the record with certain types of pathological change (119). As a result of countless research efforts directed toward this end, we may now assign the various events of the normal heart cycle, with some degree of certainty, to their respective counterparts in the electrocardiogram. The P wave is universally regarded as portraying auricular systole, while the PR interval signifies the delay of the impulse in the auricular-ventricular node. The passage of the excitation wave through the interventricular septum, papillary muscle, and the adjacent apical portions of both ventricles is represented by the Q wave while the most prominent part of R is ascribed to the activation of the mass of the ventricular walls, and the last part of the QRS to basilar excitation. Following these deflections, there is a quiescent, normally isoelectric period, the ST interval, followed by the final deflection or T wave which represents the summation of the unbalanced electrical forces in the final period of ventricular contraction. (55)

Before beginning an analysis of the electrocardiographic abnormalities resulting from coronary disease, it might be well to review the general plan of distribution of the coronary arterial supply in man. The heart is supplied by the right and left coronary arteries arising from the sinus of Valsalva just above the semilunar valves. The left is the more important from the point of view of this study being the more likely to sudden occlusion by thrombosis or embolism. It courses in the left ventricular groove to supply the anterior wall of the left ventricle, the anterior papillary muscle, and the left half of the thickness of the inter-

ventricular septum by means of its anterior descending branch. Its circumflex branch turns posteriorly in the auriculo-ventricular groove supplying the posterior wall of the left auricle and the left ventricle, the posterior papillary muscle, and the posterior portion of the septum. The right coronary artery runs along the right or posterior interventricular groove and supplies the right ventricle over its main extent. Its circumflex branch continues forward around the right side of the heart supplying the right auricle (82). This picture, while typical for the adult, is not constant throughout life since developmental changes affect the pattern. Gross* has suggested that in early life there is not much difference between the right and left coronary distribution, but with advancing years, the left coronary gradually gains the ascendancy in the number and distribution of its branches. In the aged, there is a gradual constriction in the distribution of both coronaries particularly that of the right. This may account for the more alarming symptoms in coronary disease which occurs during the period of retrogression, advancing age tending to increase the gravity of the prognosis. There is some discussion at the present time as to the exact status of anastomoses in the coronary circulation. Kahn (82) in 1922 stated, "From a clinical standpoint the coronaries may be considered as terminal. Though the capillaries may anastomose slightly when a coronary branch is occluded, the anastomoses are not sufficient to maintain adequate nutrition of the heart so as to prevent the development of symptoms." In contrast to this point of view, Wolffe

*Cited from Wolffe #195

(195) wrote as follows, "The coronaries are no longer to be considered as end arteries on account of their numerous communications especially of the deep intramuscular branches, by which collateral circulation may be maintained. Coronary disease may therefore be removed from the category of hopeless diseases and placed in a more manageable rank." Regardless of the degree of actual collateral circulation possible, no one will question the fact that the conductive system, as well as the heart muscle, depends upon the proper function of the coronary arteries for its nutrition. It is therefore quite logical to assume that any disturbances of the arterial supply of the heart's conductive system will impair its nutrition, which in turn will impair its function. According to Bohning and Katz (19) conduction is the most labile physiologic property of the heart. Since it is the first to be affected and the last to return to normal when the cardiac muscle is damaged or malnourished, impairment of the circulation should, theoretically at least, manifest itself in an alteration of the physiological electrocardiogram.

From the above description of the pattern of the coronary circulation in man, it will be evident that infarction of the ventricular musculature resulting from occlusion of one of the arteries or their main branches will be rather definitely and constantly localized anatomically in the heart. Thus occlusion may produce infarction of the anterior apical portion of the left ventricle if the anterior descending branch of the left coronary is closed, and infarction of the posterior basal portions if either the right coronary or the circumflex branch of the left is closed.

Acute myocardial infarction is almost wholly confined to the left ventricle. Although pathologically speaking, the changes produced may be of a radically different nature, nevertheless the area affected will be essentially the same whether the occlusion be sudden due to embolism or thrombosis or slow due to atheromatous changes in the vessel walls. With this in mind we may proceed to a consideration of the electrocardiographic abnormalities which are a result of alterations in the coronary blood supply.

CLINICAL DISCUSSION

Before entering into a discussion of the electrocardiographic abnormalities, it might be well to give a brief clinical classification of coronary insufficiency. The one cited below has been found to be very workable from an electrocardiographic standpoint. (20)

1. SUBACUTE CORONARY INSUFFICIENCY

A. Uncomplicated forms of myocardial infarction (thrombotic closure)

- a. Anterior
- b. Posterior

B. Complicated forms of myocardial infarction

a. Sclerotic closure

- i. Anterior
- ii. Posterior

b. Combined anterior and posterior infarction (One of the infarcts being recent)

- i. Old posterior and recent anterior
- ii. Old anterior and recent posterior

c. Multiple recent small infarctions.

2. PROGRESSIVE OR NON-PROGRESSIVE CHRONIC CORONARY INSUFFICIENCY (Coronary sclerosis without infarction.)

A. Distinctive electrocardiogram resembling that of anterior infarction

B. Distinctive electrocardiogram resembling that of posterior infarction

C. Indeterminate electrocardiogram

3. TRANSITORY (ACUTE) CORONARY INSUFFICIENCY

4. SUDDEN FATAL CORONARY INSUFFICIENCY (Thrombosis, embolism, ventricular fibrillation)

The electrocardiogram of the uncomplicated forms of recent myocardial infarction.

ANTERIOR INFARCTION

This is the most common type of recent infarction encountered clinically and is usually due to thrombosis of the descending branch of the left coronary artery on the anterior surface of the heart. (102) The resulting infarct usually involves the anterior wall of the left ventricle and adjacent septum (189) although the variability in distribution of the coronary circulation in the normal heart will alter the location slightly in individual cases. The progressive electrocardiographic changes are best understood if considered individually.

RS-T Segment

The most spectacular of the early changes involve the alterations in the RS-T segment, and take place during the stage of myocardial ischemia. (122) Instead of the normal return to the isoelectric level following the completion of the R wave, the RT segment in lead I will take its origin from a point located somewhere on the descending limb of R. The result is a plateau-like elevation of the segment of from 1 - 3 mm. in height (or larger where the maximum deflection of the QRS complex in that lead is abnormally large) which may be flat-topped, descending at a point corresponding to the apex of T in subsequent curves, or which may, in other instances, rise to form a summit at the end of the plateau before returning to the isoelectric level. This deviation is usually present within a few hours following the clinical onset of the occlusion

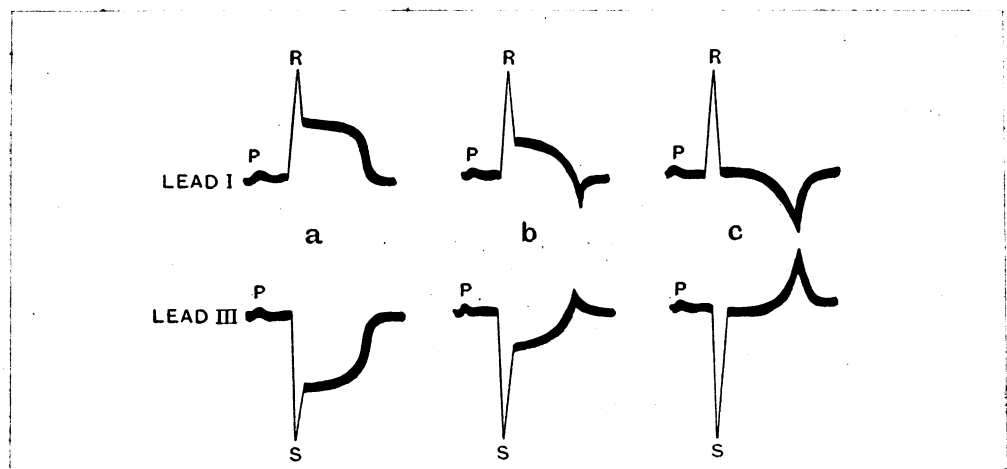


Figure 1.

Scheme illustrating the evolution of the plateau type of curve in leads I and III.

- (a) Monophasic curve, reciprocal RS-T deviation in leads I and III
- (b) Intermediate phase, T waves becoming evident.
- (c) Diphasic curve, T of opposite sign in leads I and III.

(136), but in some cases, may not appear for several days (18), and in a few instances has been reported present 24-48 hours prior to the onset of the cardiac accident. (130) The elevation at this period is described as "monophasic" (see figure 1), and it is incorrect to speak of the summit of the plateau as a T wave. The RT changes of lead 2 are similar in direction to those of lead 1 but are less marked. In direct contrast, however, are the changes in lead 3 which are directly opposite in direction, the ST segment arising from the ascending limb of S prior to the time it reaches the isoelectric line. After a variable period of time, usually from one to several weeks, the origin of the RS-T segment gradually approaches the baseline and again becomes isoelectric.

T Wave

As the RS-T segment returns to normal, a T wave begins to take shape at the point where the descending limb of the deviated limb of the RS-T segment joins the isoelectric line. This T is always developed in a direction opposite to that of the original deflection of the RS-T segment giving us a negative T1 and T2 and a positive T3. The intermediate upright T3 usually develops before the intermediate downward T1. (110) By the time the RS-T segment has again achieved an isoelectric origin, we have developed the typical "coronary T"* of Pardee (118 and 119) which arises from the

* It is the form, not the direction of the deflection, that makes the coronary T wave characteristic. Bohning and Katz (18) have pointed out that it is just as diagnostic when appearing as a positive deflection.

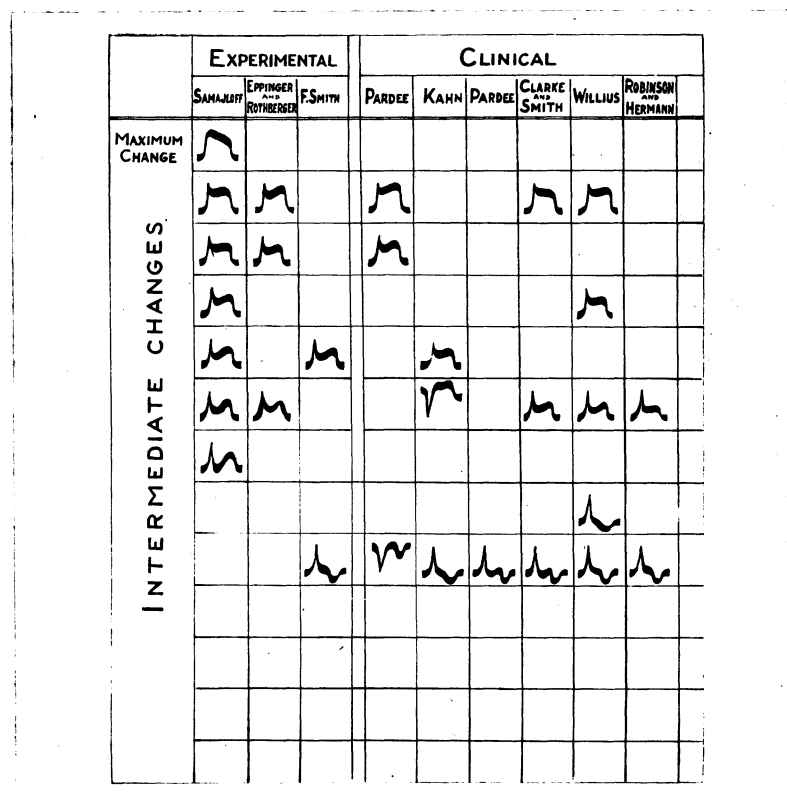


Figure 2.

Comparison as to form of the transitional curves experimentally produced by injury to the ventricular musculature with the published curves of cases of coronary occlusion.

Samajloff - No. 139.

Eppinger and Rothberger - No. 48.

F. Smith - No. 153, 154, 155.

Pardee - No. 118, 119.

Kahn - No. 82, 83.

Clarke and Smith - No. 24.

Willius - No. 174, 175, 176.

Robinson and Hermann - No. 132, 133.

curved, dome-shaped RS-T segment and which is characterized by a peaked apex and a comparatively straight returning limb. This is also identical to the "cove-plane" T wave of Rothschild, Mann, and Oppenheimer (136). The T is usually well developed at the end of two weeks, and may persist in its characteristic form from a few weeks to as long as several years. Usually there is some indication of a return to normal after three months. This change may be complete, giving us a normal upright T wave in all leads, or it may stop short leaving an inverted or flattened T1. The return to normal occurs first in lead 2, followed shortly by lead 1, while the upright T3 is last to lose its characteristic appearance finally becoming blunted or flat (122). Some evidence of residual T wave changes can usually be found after many months, and may persist permanently. During the period of transition, a simple inverted T may be the only evidence of a former occlusion. According to Barnes (8), negativity of the T wave in lead 1, arising on the isoelectric line and without any particular change in the contour of the RT segments, is enough to allow a presumptive diagnosis of coronary occlusion to be made, providing the tracing is known to have been normal in the preceding few days.

QRS Complex.

During the period following the occlusion many variable and atypical changes take place in the contour of the QRS complex in the various leads, some of which are more or less characteristic while others are entirely unpredictable. Included among the former

we have a definite* Q1 which was first described as being associated with this type of infarction by Wilson et al. (182) They characterize it as being broad and conspicuous in contrast to the absence of an initial deflection in leads 2 and 3 where the R wave is commonly followed by a deep S. Although not absolutely diagnostic of the anterior type of infarction, its presence after an acute coronary occlusion in a tracing that has not previously shown evidence of right axis deviation is very suggestive of that type of lesion. Its chief diagnostic value lies in the fact that it persists long after the ST and T wave changes have disappeared. Other less constant changes in the initial ventricular deflections include widening of the QS interval often to the point of arborization and bundle-branch block (114, 180, 37, 136, 26) distortion of the complexes with secondary R apices, M and W complexes, notching and slurring**, which Willius (174) believes indicates local disorders in myocardium affecting the conducting system, in one or more leads (14, 195, 151, 74, 189), and low voltage which will be discussed below. Among the more unusual changes is that reported by Winternitz (190) of an extensive anterior infarction in which there was a negativity of the principal QRS deflection in all leads whether Q or S. Thus while none of the changes described above may be considered in themselves diagnostic of cor-

* All Q's of at least 1 mm. which are 1/5 as large as the largest R in any lead.

** Some difference of opinion exists as to just how much notching and slurring must exist to call the tracing abnormal. Pardee (120) states that notching and slurring of the QRS in isolated derivatives can be considered normal only when found in one lead and that of relatively small excursion. If found in two, it can be considered normal only when notching occurs near the base line. It is never normal to find notching in three leads or near the peak of R in any lead of relatively large excursion.

onary occlusion when appearing in a single tracing, a sudden change in the character of the ventricular complexes in serial records is most significant.

Precordial Leads

Since Wolferth and Wood (191) first used chest leads in the diagnosis of occlusion in 1932, numerous different derivations have been suggested for use. In this discussion, I will consider only lead 4F (identical to lead CF4 if the apex is located in approximately normal position) as recommended by the committee on precordial leads of the American Heart Association. (153 and 154) Normally this lead exhibits the following picture. The P is usually small and is normally positive or diphasic, while the QRS consists of a large initial upward deflection commonly followed by a downward component of approximately equal size. The RS-T interval, while usually short and giving way rapidly to a prominent T wave, may normally show a slight positive takeoff, so that slight elevations must be interpreted with caution. Slight depressions, on the other hand, are decidedly more significant. In the anterior type of myocardial infarction, the R wave is usually small (2mm or less) and frequently absent. It is not uncommon to find a monophasic QRS of negative voltage while Bohning and Katz (20) noted a few cases exhibiting a diphasic complex with a negative initial deflection.* The RT interval shows an early elevation as in the case of standard lead I and is often horizontal in appearance so that T4 can not be made out.

* These cases are usually associated with some degree of intraventricular block.

As the RT4 returns toward the isoelectric or normally positive level and regains its upward declivity, a diphasic and then a negative T wave appears which waxes to great height before waning and disappearing. (20) The fully developed negative T wave is characteristically sharply peaked and is often of abnormal depth (over 10 mm)* In conjunction with the above changes, there often occurs notching of the initial ventricular deflections, and the P wave occasionally becomes diphasic if previously positive. That all investigators are not in exact accordance as regards the way in which this lead should be interpreted is illustrated by the fact that Goldbloom (147) does not consider a diphasic T as abnormal, and he entirely ignores ST4 deviations. Since the RS-T and T wave changes may occur sooner and last longer in this lead than in the standard derivations, pathology is often detected here which might otherwise have escaped unnoticed if only the limb leads had been taken. (173) Here again it might be well to emphasize the importance of numerous, well-timed serial tracings in a case of suspected coronary occlusion since it is the rapidly changing form of the complexes rather than any single configuration itself which, in the last analysis, makes the diagnosis certain.

POSTERIOR INFARCTION

By the term posterior infarction, we refer to those infarcts on the posterior surface of the left ventricle near the base due to

* Willcox and Livibond (173) consider a T4 over 10 mm in amplitude whether positive or negative as suggestive of localized apical infarction.

A large T4 is usually associated with large upright T2 and T3 (166)

occlusion of the right coronary artery especially of the posterior branch (165). In this form of occlusion, the changes in the electrocardiogram are produced by changes of potential exactly opposite to those acting in the case of anterior occlusion. The RS-T and T changes, therefore, are exactly opposite to those discussed above, consisting of an elevation in leads 2 and 3 and a depression in lead 1 with intermediate T waves developing into final deflections in the opposite direction. A large Q wave in lead 3 is much more liable to develop here than is the prominent Q1 of the anterior type. The return toward the normal electrocardiogram follows the same sequence as described above. As a rule the changes in the precordial leads are less spectacular and less characteristic in this form of occlusion. In the early stages there is usually a depression of the RS-T segment, but the deviation is not so marked as in the anterior type. In the period when the segment is depressed it tends to become horizontal with or without a downward bowing, and its termination is to be more clearly differentiated than the normal upward slopping. In the series of Bohning and Katz (20),

-
- * Much has been written concerning the large Q3 characteristically appearing in this form of infarction. Although many theories have been advanced as to its cause, the predominant belief is that it represents posterior septal involvement (53). The criteria for the diagnosis of a large Q3 are as follows. (121)
1. Excursion must exceed 25% of the greatest excursion of R in any lead.
 2. The tracings must show left or normal axis deviation.
 3. All records must show an initial downward deflection in lead 3 followed by an R with no S deflection.
 4. Tracings must be omitted which show M or W complexes.
- Durant (43) modifies the criteria by allowing those tracings to be considered that show an S deflection and also those with slight notching of the downstroke of Q (the W complex of Pardee)

horizontal ST4 often occurred without depression, and they considered this a characteristic finding in recent posterior infarction. Following the return of the segment to the isoelectric, R4 becomes large and T4 is usually strongly positive. Generally speaking, lead 4 is of less value in this type of infarction since it is less likely to show changes in the absence of changes in the limb leads. In both varieties of infarction, the changes in the initial ventricular deflections in the chest leads are the most permanent of all the abnormalities outlasting the high voltage T waves. Thus the absence of R4 in the anterior lesion and the large R4 in the posterior lesion may be the only residual changes when the first tracing is taken several months following the cardiac accident.

There now remain to be discussed several electrocardiographic abnormalities which appear in and are more or less characteristic of both types of myocardial infarction but also are to be found associated with various other varieties of coronary insufficiency. They are as follows:

P Wave Changes.

In 1933, Masters (106) called attention to certain changes in the auricular wave which he believed were characteristic of coronary occlusion. He examined a series of forty cases and found definite P changes in 80%. These alterations consisted for the most part of increased amplitude with occasional notching and widening. They occurred most frequently in lead 2 with leads 1 and 3 following in that order. The P wave changes are most manifest, he declared, during the first few days of acute illness when cyanosis, congestion

of the lungs, enlarged liver, and severe dyspnea or orthopnea are present. He considered the changes to be due to auricular dilatation. Probably no great significance can be attached to this finding since in relatively few of the cases of acute occlusion do we have control tracings with which to compare and thus detect relatively small increases in the size of the auricular complex.

Arrhythmias

In surveying the literature, one finds evidence of every imaginable type of disordered rhythm following acute coronary occlusion. These include premature contractions of both auricular and ventricular origin (132, 156), auricular fibrillation (112, 151) and flutter (126) which may be paroxysmal in nature (3), simple sinus arrhythmia (175), ventricular tachycardia (132) and ventricular fibrillation which may occur as a terminal event (see below). Puddu (126) found auricular fibrillation to occur much more often than flutter. This is the result, oftentimes, he believes, of occlusion of the right coronary artery with a shutting off of the blood supply to a large portion of the right auricle and the sinus node. In the case of occlusion of the left coronary artery, he explains the phenomenon on the basis of a reflex which originates in the injured portion of the heart muscle or by overwork which throws a burden on the auricles in all cases of myocardial infarction. In almost all cases of intermittent flutter and fibrillation, the paroxysms are introduced by auricular extrasystoles.

Disturbances of Conduction

Beside frequent examples of block of the arborization and

bundle-branch variety mentioned above (175, 180, 194, 179) which are considered by Bohning and Katz (20) to be indicative of septal involvement,* there are also to be encountered, following attacks of occlusion, various manifestations which result from the shutting off of the blood supply to the AV and SA nodes. These range all the way from the so-called "wandering pacemaker" (86) due to disturbance of circulation around the SA node (supplied by the ramus ostii cavi superioris which may be a branch of either of the two main coronaries (67) through disturbances of AV conduction (112, 77) to complete heart block (112, 86, 179, 158) resulting from alterations in the circulatory supply to the AV node (supplied by the fourth from the last main posterior descending branch of the right coronary which gives off the ramus septi fibrosi to the node (67)).** The great majority of cases of complete heart block occurring after acute uncomplicated infarction are associated with closure of the right coronary artery.

Low Voltage

Decreased amplitude of the main deflections is a frequent finding in both of the main uncomplicated types of coronary occlusion.

-
- * Not all of the authors cases exhibiting septal involvement show evidence of intraventricular block.
 - ** Kugel (91) found a large vessel, the arteria anastomatica auricularis magna, a branch of the left coronary artery which anastomoses freely with the artery of the AV node. Because of this he considers that the node will receive insufficient blood supply only on rare occasions such as in.
 1. Acute right coronary thrombosis plus sclerotic narrowing of the opposite coronary or its branches - or
 2. Sclerotic narrowing of the left coronary and temporary insufficiency of the blood supply here by reason of congestive failure or extreme physical exertion. He considers this the most satisfactory explanation of transient block.

Its presence has been reported by many investigators (155, 161, 151, 33, 74, 147, 185) who conclude that not only does a reduction in the size of the ventricular complexes occur in occlusion but that coronary disease is one of the main causes of low voltage, accounting for 76% of the cases reported by Steuer.* The low voltage may be of a transient nature and may disappear simultaneously with signs of clinical recovery of the patient. It is considered an indication of poor prognosis when the fall in amplitude is marked and persists at a low level or is constantly progressive beyond the first week or ten days. Winternitz (190) believes that the cause for the lowered voltage may be found in the disturbed muscular balance. Here again we see emphasized the advantage of serial tracings over the single record which can give no indication of the tendency or rate of progression of voltage changes.

Axis Changes

Only during the last few years of electrocardiographic investigation has emphasis been placed on the care which must be exercised in the interpretation of axis deviation in tracings. No longer can the term axis deviation be considered synonymous with ventricular preponderance or hypertrophy since many other conditions, including changes in position of the heart as in pregnancy (147), will alter the potential difference in respect to various planes of the heart. First notice of a prompt shift to the left following thrombosis was made by Pardee (118) who considered it due to involvement of the

* This series took into consideration coronary sclerosis as well as acute occlusion.

circulation of the septum. Since then much research has been done on the subject (175, 14, 112, 151, 88) by many investigators who stressed the sudden change from left to right and vice versa following arterial occlusion. Van Nieuwenhuizen and Hartog (167) stressed changes in the electrical axis following occlusion which might be first evidenced in the chest leads* due to a dilatation of the left half of the heart beginning in a sagittal plane, and which later would be manifest in the limb leads. They disagree with Nuzum and Elliot (113) who attribute all left preponderance curves to coronary changes since they found left axis deviation occurring so consistently in young people with chronic nephritis that would be very unlikely to show coronary damage. Left axis deviation, wrote Van Nieuwenhuizen and Hartog, "is generally not seen when no dilatation exists. Absence of left axis deviation in hypertension which has brought about a heart dilatation is in our opinion caused by other factors which conceal the deviation in these cases". Bohning and Katz (20) found left axis shift with all types of infarction, but marked deviation associated with preponderance of the left ventricle was more common with anterior infarction. They found right axis shift only infrequently and then always associated with the anterior variety of infarction. Occasionally both right and left shifts

* Method of detecting axis deviation from the chest leads as outlined by these authors is as follows. Normally R is never under 2 mm. and S usually does not exceed 17 mm. Normal S:R ratio is 2:1, showing normal extremes of from one to eight. In the left axis deviation there is an increase in the height of S and a disappearance or decrease in the depth of R so that the ratio values go to 10:1 or more. Curves with anterior coronary infarction and those with bundle-branch or arborization block must be excluded.

were transitory in nature. All authors agree that sudden axis shifts such as may be encountered only on well-timed serial tracings are of considerable significance, but that there is no single axis pattern that can be considered typical of any particular form of occlusion.

Optimum Time for Taking Tracings in Uncomplicated Infarction.

In addition to the need for serial tracings much emphasis has been placed upon the proper timing of the records in order that minor changes in the form of the complexes, which may be the only evidence of an occlusion, will not escape unnoticed. Richter (130) recommends the following intervals as most satisfactory:

- a. As soon as possible after the accident.
- b. The next day following the accident.
- c. 48 hours following the second.
- d. Weekly intervals for as long as necessary.

A brief discussion of the events taking place in the heart muscle following occlusion will serve to establish the logic of the above schedule. The early characteristic ST segment and T wave changes are essentially transient phenomena (189) and can not be ascribed to the disappearance of the electrical forces normally produced by the necrotic muscle. On the other hand they can not be expected to arise directly within the dead tissue which can, of course, produce no electrical effects. It is clear, however, that these changes together with the QRS alterations that follow or accompany them, must have their origin in events that occur with the muscle zone that normally receives its main blood supply through the obstructed vessel. It seems logical that the RT displacement following occlusion is due to the disappearance during systole of a current of injury flowing

at the boundary of the infarcted area (42, 40). The T wave changes appear because of the prolongation of the duration of the excited state in the muscle that gives rise to the current of injury, and the QRS changes, which are more permanent, to the absence of electrical forces normally produced by the infarcted muscle (189). Thus we should expect to find ST segment changes occurring during the first week or ten days throughout the period of infarction and necrosis, the T wave changes following later during the period of organization and repair (119), and the permanent QRS changes persisting during the recovery phase as a memorial to the scar remaining in the myocardium.

Prognosis on the Basis of the Electrocardiograph.

For many years, it has been a well known fact that the case of posterior infarction carried a much better prognosis than does one of the anterior variety. In considering the significance of this fact, it must be remembered that clinical series show the anterior variety to be the more common (13, 102) while pathologists report about an equal incidence of the two types. Van Nieuwenhuizen and Hartog believe that this may be the result of the fact that the anterior variety results more commonly from acute closure while the posterior type is found more often following a gradual narrowing of the lumen of the vessel. Thus we see that on the basis of localization alone, we have grounds for electrocardiographic prognosis. Moreover, considered as a group, the occlusions showing a definite and typical picture (i. e. either anterior or posterior) seem to have a better prognosis than the indeterminate type with small,

slowly developing, atypical changes (50). This may be due to the fact that the infarction in the former case is single and not large. As to prognosis within a given class (i. e. either anterior or posterior) the facts are less definite. Bloom and Cashion (16) believe that if, after thrombosis, the electrocardiograph reveals a spiked QRS with no appreciable widening and above five millimeters in voltage, the chances of recovery from the attack are excellent. If, however, the tracings reveal notched or slurred QRS complexes and/or a voltage of five millimeters or less in one or more leads, the patient will probably succumb or have irreparable heart damage. In concluding they state, "To consider these findings as absolutely prognostic phenomena would be absurd, but their influence as additional (to clinical) prognostic data should not be disregarded."

Atypical Pictures

Although many of the complicating factors that tend to confuse the electrocardiographic picture will be left to be discussed in another section, it must be remembered that even uncomplicated cases of simple anterior and posterior infarction will not adhere to exactly the same pattern. Wood et al. (197) list the factors which tend to alter the pattern in individual cases as follows:

1. Anatomic variations in the coronaries in various subjects are responsible for many unexpected electrocardiographic results.
2. The type of electrocardiograph depends upon the relative position of the heart with its infarct and the electrodes. Changes in the position of the heart in the experimental animal gives changes in a given infarct. Considering the various size, shape, and position of heart and axis an infarct in a given position will vary in its electrocardiographic manifestations in minor ways.

3. The electrocardiographic abnormalities in acute occlusion are probably dependent upon the exact size and location (with reference to the electrodes) of the ischemic area of the heart. The area of ischemia may be different from the area of infarction found at necropsy and varies from time to time depending upon.
 - a. The efficiency of the general circulation.
 - b. The condition of the adjacent coronary circulation.
4. As pointed out above, marked variations of the ST segment are caused by injured but living muscle. When this muscle dies the deviation disappears. If, therefore, the heart is the seat of a large infarct in which the major portion of the involved heart muscle is dead, only small RS-T deviations might be recorded. After several days, the obtainable RS-T deviations are probably produced by muscle situated at the periphery of the lesion, rather than by the main infarcted muscle mass. Thus if no tracing is taken shortly after the onset (as recommended above) we can not say that the recorded deviations of the RS-T interval were caused by the ischemic heart muscle located as the pathological lesion is located. The main muscle mass may be dead and incapable of giving rise to RS-T deviations. For this reason experimental lesions made with a cautery (35) must probably be interpreted with caution especially if the lesions are large.
5. Previous electrocardiographic abnormalities will alter the final picture resulting after an acute occlusion.
6. Other factors less well understood (130), such as
 - a. Chemical and physical reaction of the blood and tissues.
 - b. Platelet count.

Finally, before leaving the discussion of uncomplicated infarction, it is only fair to state that in some cases no demonstrable abnormalities of the electrocardiogram will be encountered. The incidence of cases in which positive findings are obtainable have been much increased both by the employment of chest leads and the utilization of serial tracings, but there still remain some records which must be called normal according to existing standards. On the other hand, according to Wilson et al. (189) "A series of electrocardio-

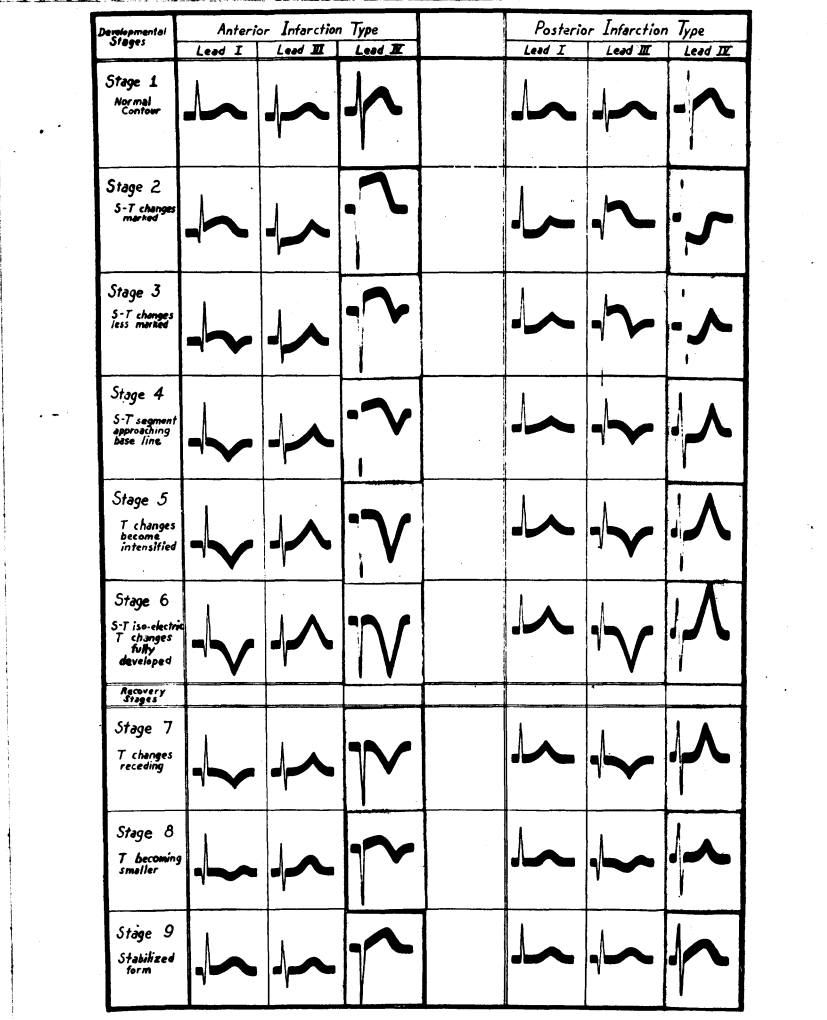


Figure 3.

Diagrammatic illustration of the classic type of changes usually found in leads 1, 3, and 4 (CF_4) in the stages of development of and recovery from uncomplicated infarctions of the anterior and of the posterior wall due to sudden thrombotic closures.

grams that are normal in all respects and have been taken in the period immediately succeeding the onset of the suspected infarction should not be wholly disregarded unless the symptoms and physical signs pointing to this condition are unequivocal."

This brings us at this time to a discussion of the complicated forms of myocardial infarction.

SCLEROTIC CLOSURE

Under this heading are considered those infarctions that result from the more or less slow narrowing of the lumen due to the formation of atheromatous plaques in the vessel walls or from marked coronary sclerosis with or without congestive heart failure. Anatomically they may, as in the case of the uncomplicated forms, be classified into anterior and posterior varieties according to the vessel occluded, but for several reasons, the electrocardiographic picture which they present is often quite different from the uncomplicated forms. The factors most responsible for this difference are the gradual, instead of sudden, onset of the occlusion with consequently more time for the establishment of collateral circulation to the area involved, and the condition of the remaining coronary circulation, for it stands to reason that an extensive sclerotic process in one coronary artery will be accompanied by sclerosis of an almost if not equal degree in the remaining coronary circulation. Thus we find electrocardiographic patterns that vary considerably from the classic anterior and posterior types in individual cases. For example the RS-T segments may be depressed in all three of the standard leads although they are rarely elevated in all. Bohning and Katz (20)

found ST3 elevation of all cases of closure due to sclerotic plaques coming to autopsy whether the vessel occluded was the right or left coronary. The T wave pattern may be bizarre, a negative T3 occurring occasionally in cases of anterior infarction while negative coronary T's have been reported in all the standard leads following an occlusion of this type (20). The most common variations from the classic T changes are found in the anterior type of infarction from sclerotic plaques. Q changes are also far less constant and more unreliable. While a large Q1 is commonly seen with uncomplicated sudden anterior closure, it is rarely seen with occlusion resulting from plaques; however, Bohning and Katz (19) report several cases of sclerotic occlusion in which a large Q wave was present in all leads. The arrhythmias, delays in conduction, disturbances in voltage, and axis deviation seen in the previous type also complicate this form of closure. The findings in the thoracic leads are also not definitely predictable, Van Nieuwenhuizen and Hartog having described a picture which they consider typical for the posterior sclerotic occlusion (found much more commonly in their series than anterior infarction which they believe is due more often to sudden closure). Here the initial deflection is downward followed by a tall R* wave which is usually notched and an ST segment whose takeoff is depressed, that is concave in appearance, and, "into which creeps a positive T wave." The small Q, "may be missing and may occur in other types so that it may be valued only in connection with other deflection." The most characteristic

* Bohning and Katz (20) believe that R is more often small when closure is due to plaques.

point is the depressed and concave course of the RS-T segment. In some cases the ST segment may be flat but keeps on dropping too low and runs below the isoelectric level. The associated pathology in other leads is usually as follows:

- a. Lead 1 shows an absolutely or relatively too deep S and low T.
- b. Lead 2 shows a too broad ventricular complex, notches in the R or S, and a too deep S wave. The PR interval is always prolonged.
- c. Lead 3 shows a deep S wave, notches after the S wave, a flat, diphasic, or negative T and sometimes a Pardee Q. The convex ST segment usually has a high takeoff and W or M shaped ventricular complexes are commonly present.

Proof that this lesion is of the posterior variety is offered as follows:

- a. There is confirmatory evidence in lead 3. (Barnes and Whitten 11, Wilson et al. 189)
- b. The ventricular complexes are broad, having approximately the shape of an arborization block, and the atrioventricular conduction time is prolonged beyond the maximal. Both of these may be ascribed to alterations in the circulation of the posterior coronary artery.
- c. Absence of the deviations characteristic of coronary occlusion in lead 1.
- d. About the same form is sometimes seen in ventricular extrasystoles in the chest leads when ventricular systoles of the left type exist in leads one and three.

Bohning and Katz (20) state that the QRS4 is less liable to be entirely negative in this type of infarction than in that resulting from sudden closure but is usually mainly so. In conclusion it might be said that the electrocardiogram of the case of infarction

resulting from closure due to plaques or sclerotic processes resembles the picture due to infarction from sudden occlusion in its principal features and general tendencies, but differs in many essential minor details, not the least important of which is the time relationship of the progressive alterations.

COMBINED INFARCTIONS

According to Sprague and Orgain (158), "Acute coronary thrombosis with closure limited to a single artery or branch of an artery is relatively rare on the autopsy table." If this statement is true, it must follow that the combined forms of infarction should receive serious consideration from an electrocardiographic standpoint. Although so much variation results, depending upon the anatomical locations of various infarcts in the same heart and the time interval occurring between the various occlusions, that the description of a given picture which will hold for any large number of cases is impossible, certain generalities may be cited. Because of the time relationships of the various complex changes, it must be obvious that when two infarctions occur with an interval between, the final picture will include the acute (RS-T) changes of the more recent infarct and the residual (T and initial deflection) changes of the primary occlusion. Thus with a recent posterior infarct superimposed on a previous anterior, we might expect to find a large Q1 and possibly a negative T1 indicative of the initial accident, together with an elevated ST 2 and 3 resulting from the more recent process (20). Winternitz (190) describes a syndrome which he considers characteristic of dual infarction which involves a shrinking of all of the

main deflections while the smaller ones remain unchanged (confirmed by Gagu 60). Wolferth described several cases of combined anterior and posterior infarction in which the ST segments were elevated in all leads (maximum in lead 2) and others in which they were depressed in all leads. In a consideration of the precordial leads, Willcox and Loviband (173) describe a large R suggestive of posterior infarction combined with an absent S indicative of possible anterior infarction, while Bohning and Katz (20) describe the QRS4 complex as mainly negative in cases of combined infarction. Wood et. al. (197) published records of a case of acute anterior infarction with residual large Q3 and inverted T 2 and 3 suggesting a previous anterior infarction. These same authors also described a case of acute anterior infarction at the extreme apex which had been the site of a previous extensive infarction, the records showing no RS-T deviations in any of the leads. Priest and Saphir (130) showed reports of a patient who had coronary thrombosis without infarction who later developed infarction in the area supplied by the original occluded vessel following the occlusion of vessels supplying collateral circulation to this part.

Multiple small infarctions will also produce atypical and bizarre records. If they occur in the same location one will find repeated series of acute changes superimposed upon the residual changes from the previous infarctions. Smith (156) described a case in which there were several attacks of severe precordial pain. Progressive extensions of the T deflections in serial electrocardiograms were thought to indicate further cardiac damage possibly by the

nipping of small arterial twigs (56). Thus we see that every case represents an individual problem which must be evaluated using the clinical data to substantiate the serial electrocardiographic records. It has been stated, and it seems logical to assume, that this type of case carries with it a higher mortality than do the uncomplicated single myocardial infarctions. It has also been pointed out that these forms are more often complicated by intraventricular block possibly on the basis of more frequent involvement of the septum due to the presence of more than one infarct(20).

PROGRESSIVE AND NON-PROGRESSIVE CHRONIC CORONARY INSUFFICIENCY

Under this heading are included all types of non-regressive coronary insufficiency without cardiac infarction. Some varieties of chronic coronary insufficiency whether progressive or non-progressive have tracings resembling those of myocardial infarction except that the serial curves do not follow the course typical for cases of healing infarction. Such a course might therefore be considered characteristic of the changes due to local ischemia while its absence might be considered indicative of generalized ischemia, since it must be remembered that the electrocardiogram does not depict the fibrosis or even the infarction but rather the presence of localized or generalized anemia of the living muscle cells (141, 20). In the non-progressive form, the four lead electrocardiogram shows abnormalities which are practically stationary over long periods of time, months or years, since the collateral circulation is able, either because the pathological processes are developing slowly or not at all or because the impairment is not too far advanced

to keep pace with the pathologic processes. In the progressive type, on the other hand, the electrocardiograph shows abnormalities which progress slowly over periods of months or years or more. Here the collateral circulation can not keep pace with the pathological processes either because the anatomical changes are too far advanced or because cardiac congestive failure is occurring (19). The typical coronary T was first shown by Pardee (119) to be a typical finding in coronary sclerosis. That it should occur frequently in cases of chronic narrowing is not surprising when one remembers that its pathological basis is not found in the primary anemia of coronary occlusion but in the secondary action of repair about the anemia area. Thus its appearance is not dependent upon infarction and it appears often in those patients with a slowly progressive narrowing of an arterial branch with a local area of chronic ischemia and a surrounding secondary reaction of repair. It may disappear in later records where collateral anastomoses have taken over the blood supply of the damaged area or when this has been replaced by fibrous tissue. It is significant of the specificity of the coronary T wave that it does not appear in cases of chronic fibrous pericarditis or with chronic valvular disease (119).* Pardee believes its association with definite atheromatous changes in the vessel walls has definite prognostic significance and states, "When it (the coronary T) is present the possibility of sudden death from cardiac infarction can not be denied." Beside the typical coronary T, many investigators report a simple inversion of T as a constant finding. Strong and

* Later investigations have shown that this statement may not be entirely true. (See section on differential diagnosis.)

Caverhill (164) found T wave changes in 53% of their series but did not differentiate the types. Nathanson (112) found simple T inversion as the most frequent abnormality and described only 25% of these as coronary T's. He considers the simple inverted T equally as significant of coronary pathology, however, although he does not consider it specific. Strong and Caverhill (164) found deviation of the RT or ST segments in 61%. Gager (60) found 60% of his series of coronary sclerosis showed a "saddle" type of ST deformity, "an abnormality", he states, "which in its pure form is found almost exclusively in coronary sclerosis". He describes the deviation as a curving ST with an upward concavity which in their records took its origin somewhat above the isoelectric line, from the descending broadening limb of the R wave. The seat of the "saddle" may or may not sag below the isoelectric line and usually merges in a broad upright T. It occurred with about the same frequency in any one of the limb leads and frequently was found in all three. Other abnormalities of the initial ventricular deflections include large Q waves (16% of the series of Gager - most were large Q3) frequent minor notching and slurring of QRS (48% of the series of Nathanson) and bundle-branch block (10% of the series of Nathanson.) Also frequently occurring are arrhythmias, of which the most common are extra-systoles and auricular fibrillation, low voltage, left axis deviation and prolonged atrioventricular conduction time. Complete heart block is a rather rare accompaniment. Gager (60) noted the association of a low erect T wave, an erect T3 wave, and a definite S2 wave in a group of patients of whom four showed no left axis deviation.

The precordial leads have been found to be extremely valuable in the diagnosis of sclerosis, often showing changes when none exist in the limb leads. Patients with coronary sclerosis having clinical evidence of myocardial incompetence showed more abnormalities in lead 4 than patients without. The major types of deflections in the precordial leads may be listed as follows:

QRS Configurations.

1. Negative QRS4 type - (42% of the series of Bohning and Katz (19). In this type the QRS complex is mainly or entirely negative. R is usually present but very small. The T wave is usually positive and normal but sometimes is diphasic and occasionally over 10 mm.
2. Positive QRS4 type - (8% of the series of Bohning and Katz (19). Here the QRS4 complex is mainly or entirely positive. In about 50% of the times it is diphasic with a large R M and W forms are occasionally seen. T is usually normal but may be negative or diphasic.

Neither of the above two types are constantly associated with one of the QT types described in standard leads. (Section on uncomplicated infarction). The ST segment is usually within normal limits.

T Wave Configurations.

1. Negative or Diphasic T4 - (25% of the series of Bohning and Katz (19). When of a diphasic character the first wave is usually positive. The QRS is usually diphasic and not abnormal, but a few show negative QRS4 types and rarely a positive QRS4 is found. The RS-T segment is usually within normal limits but a few show deviations either positive or negative.
2. Strongly Postive T4 - (7% of the series of Bohning and Katz (19). This variety of T wave is usually associated with the negative QRS4 complex. ST is always positive, but in only a few cases is it outside of normal limits. When QRS4 is diphasic, it is occasionally large or unusually small or the mirror image of the normal so that the first phase is negative and followed by a positive phase of equal magnitude.

One can not help but realize here that a differential diagnosis from certain phases of the recovery process in acute infarction can be made electrocardiographically only with serial tracings. Once the alteration in the ST segment disappears and only the alteration in T remains, it becomes difficult to tell from a single record whether the tracing shows:

1. An early state of recovery after infarction in which T is waxing.
2. A late stage of recovery in which T is waning.
3. A chronic coronary insufficiency without infarction.
4. An old infarction.

The clinical history will of course help to differentiate between 3 and 4 while the electrocardiograph will differentiate between 1 and 2 and will also separate 1 and 2 from 3 and 4.

TRANSITORY ACUTE CORONARY INSUFFICIENCY.

Transitory acute coronary insufficiency may occur in the presence of other coronary disease as a result of various causes such as excessive effort and emotional crises. It may also appear in very rapid or irregular heart action, infarction of the kidneys, spleen or lungs, during congestive heart failure, or as a result of syphilitic involvement at the mouth of the coronaries. The transitory insufficiency does not result in permanent damage to the myocardium but causes temporary electrocardiographic deformity which may last a few minutes to several hours, after which there is a return to the pre-existing contour. Attacks such as these may be accompanied by definite anginal pain or by only vague symptoms similar to those which may occur with paroxysmal cardiac dyspnea. The changes in the elec-

trocardiogram are brought about by alterations in the condition of the coronary blood supply which make it inadequate for the work the heart is doing, either because the former declines suddenly or the latter increases abruptly (19).

The first series of records taken during anginal attacks were those reported by Siegel and Feil in 1928 (150). Control records were taken prior to the attacks which were compared to records taken at the height of the pain. The most marked changes were in the ST segment which was consistently depressed, most often in leads 1 and 2. The T wave changes were less consistent, T sometimes changing from the inverted to the diphasic form, in other instances showing definite alterations in amplitude. R usually remained constant, but on one occasion showed a definite diminution in amplitude in lead 2. The PR and QS intervals remained normal. One atypical case showed no changes at all during the pain while another showed definite diminution of the amplitude of the previously inverted T1 and T2 after the pain had subsided but not during the attack itself. They concluded that, "Changes in the ST portion of the electrocardiogram of patients studied probably represents transient vascular changes in the heart muscle."*

Bousfield (22) reported a case of aortic insufficiency

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- * The authors made an interesting study of other factors that might have altered the contour of the ST segment.
1. Vagal tone - (Carotid sinus pressure) T3 inversion increased from 3 - 5 mm. Rate slowed. No other changes.
 2. Cold water - Pressure - Pain - Caused no changes in the PR int., duration of R or QRS, or in the T waves.
 3. Respiration - Changed height of the initial ventricular deflection but caused no change in ST or T.

which exhibited a transient bundle-branch block during the period of pain which completely disappeared following the attack. He decided that the most striking electrocardiographic changes occur in patients with anginal attacks where there is evidence of some myocardial involvement in the control tracing (22).

Cowan and Ritchie (34) reported similar changes in the T contour with additional evidence of prolongation of the QS interval and alteration of the ventricular preponderance. With cessation of the attacks all of their records returned to the normal.

Levine et al. (96) made exhaustive studies of patients in whom the anginal attack was produced by injections of epinephrine. They found all of their records taken during the attack showing changes in the "normal" direction (i. e. the same type of change as in the controls, differing from the latter in the degree but not in the direction of the change.)

Wood and Wolferth (198) published an extensive study of anginal patients, in some of whom the attacks were spontaneous, and in others in which they were induced. Controls were run to rule out changes due to exercise alone and to alterations in the blood pressure and pulse rate. By this means, they discovered that over half of the changes seen such as the development of a transient right axis deviation and the tendency toward an increased positivity of the T wave, whether negative or upright in the controls, were due to exercise. Other changes which were "specifically" ascribed to the attack included:

1. Tendency toward T wave negativity. Definite coronary T3 appeared in some of these cases.
2. Depression of the ST segments usually in lead 1 alone or in leads 2 and 3 in combination.

Alterations of the ventricular complexes were most marked when the pain was at its height and usually disappeared a minute or two after the pain had subsided. In a few cases, the most marked changes were noted after the pain had ceased. Since the same percentage of the patients with mild attacks (50%) showed changes as did those with severe attacks, they concluded that the intensity of the pain did not bear a definite relationship to the changes, a conclusion born out by the later observations of Katz and Landt (86). In commenting on the epinephrine inductions of Levine cited above, they conclude, "Epinephrine may dilate the coronary vessels and thereby mask the true nature of the electrocardiographic changes due to the angina pectoris itself."

Considerable additional information on this problem was contributed by Katz and Landt (86) who suggested a series of standardized exercises* with which to induce anginal attacks in susceptible individuals for electrocardiographic study. Their observations were made using chest leads as well as the standard deriviations. In the standard leads, they noted axis shift to the right in a few instances, changes in the T waves which usually occurred in the

* The exercise consisted of raising a three pound dumbell in each hand by extending the arms fully at right angles to the trunk a total of 60 times in three minutes. The attacks induced in this manner were mild and resembled the spontaneous variety since the effort expenditure is limited due to the development of fatigue in the arms. Exercise was stopped in any case where generalized fatigue, dyspnea, cyanosis, or anginal pain appeared before the three minutes were up.

opposite direction to the major deflections of the QRS complex, and depression of the ST segment, although on one or two occasions when the major deflection of QRS was negative in a given derivation, ST was elevated in this lead. Changes in lead 4 included downward "angular" deviation of the ST segment making the angle between the ST and the ascending limb of T more acute, and increased positivity of the T waves. In a few instances, the ST segment was depressed. Twenty-five percent of their series showed no electrocardiographic changes during the attack and in only 12% were abnormal changes limited to the fourth lead.

That the electrocardiograph may be of definite prognostic value in angina is suggested by Collins (32) who found that the highest mortality occurred in that group of patients who showed inverted T waves, signs of intraventricular or atrioventricular block, or low voltage during the attack. Moreover, Wood and Wolferth (198) found that all of their patients showing "specific" changes during an attack died within a relatively short time.

Thus we may conclude that the changes taking place during acute coronary insufficiency are morphologically similar to those seen in other types of coronary disease differing primarily only in duration, being relatively transient.* It is of considerable importance in evaluating these tracings to rule out abnormalities present in the control tracings, since in many cases, an acute coronary

* Blumgart et. al. (17) have shown by experimental work that temporary cardiac ischemia may result in lasting electrocardiographic changes in the absence of any discernable histologic evidence of myocardial necrosis.

insufficiency is superimposed upon a chronic milder coronary insufficiency, which in itself will produce electrocardiographic alterations and thus serve to confuse the picture. In addition to this, the changes due to exercise or drug administration, when either of these two methods are used to induce the attack, must be carefully evaluated before those abnormalities which appear on the tracing during the attack of pain can definitely be said to be solely the result of the angina pectoris.

SUDDEN FATAL CORONARY INSUFFICIENCY.

This condition may justifiably be dismissed here without further discussion since it is the general rule for these cases to terminate fatally in ventricular fibrillation (199, 43, 110) before sufficient time has elapsed in which recourse to electrocardiographic analysis can be made. The experimental work of Blumgart et al. (17) lead these investigators to conclude that the occurrence of ventricular fibrillation in cardiac infarction and in other conditions in which the heart is irreparably damaged may be due to the presence of zones of impaired nutrition in proximity to areas of complete ischemia.

DIFFERENTIAL DIAGNOSIS

Ever since the electrocardiogram was first used in the diagnosis of coronary artery disease investigators have been aware of the fact that no single finding or group of findings could be considered absolutely pathognomonic, but rather that it is the progressive serial changes viewed as a whole that makes the characteristic picture of coronary involvement. For this reason, it was recognized early that tracings taken during the course of many other disease entities will closely simulate a single coronary record in one or more of its more characteristic features. A study of these interfering factors is most easily made by taking up again individually the various abnormalities encountered in coronary artery disease and discussing the other pathological conditions which may produce them.

A. The Simple Inverted T Wave.

Simple inversion of the T wave in one or more leads such as is observed often in various types of coronary insufficiency is also encountered in many other conditions among which have been listed:

1. Experimentally cooling the apex of the heart - delays or prolongs the activity at the apex so that the record of the final activity at the base is interfered with (119, 183)
2. Hypertension - especially when associated with left ventricular preponderance. (112) (See discussion at end of this section)

3. Valvular disease. (112)
 4. Following severe diphtheria. (112)
 5. Myxedema. (74, 112)
 6. Factors causing left ventricular strain - resulting in T wave inversion in leads 1 and 2 as: (179)
 - a. Hypertension.
 - b. Aortic stenosis.
 - c. Aortic insufficiency.
 7. Digitalis - This is a common cause for an inverted or diphasic T wave that often causes confusion when the history is incomplete. (112, 74)
 8. Vagus stimulation. (112)
 9. Pneumonia and other toxic states. (74)
 10. Uremia. (74, 158)
 11. Factors causing a change in position of the heart as:
 - a. High diaphragm. (74)
 - b. Pregnancy.
 12. Various factors causing right ventricular strain (179)
- Leads 2 and 3.
- a. Arteriosclerosis of the pulmonary artery.
 - b. Marked pulmonary emphysema or fibrosis.
 - c. Pure mitral stenosis.

13. Pericarditis (158)
14. Rheumatic carditis. (158)
15. Certain rare unexplained diseases. (158)

Nathanson sums up the situation as follows, " In the absence of enlargement and congestive heart failure T wave inversion is practically pathognomonic of coronary sclerosis."

B. The Coronary T Wave

The appearance of this type of wave in conditions other than coronary disease is even more confusing than the presence of the simple inverted T wave. It has been found in:

1. Valvular lesions of endocarditis (130) both
 - a. Rheumatic, and
 - b. Syphilitic.

Berman and Mason (14) reported that most of their syphilitic patients suffered from anginal attacks while Pardee (119) stated that his were free from pain. Both believe that involvement of the mouths of the coronaries figured in the development of the abnormal wave.

2. Diabetes with vascular degeneration. - In these cases damage of the left ventricular muscle or the left coronary artery by an inflammatory process or by an infarction following embolism might easily have occurred. (14)

3. Fulminating purpura hemorrhagica - This condition may have been complicated by hemorrhage into the myocardium or epicardium. (14)

4. Pernicious anemia. (14)
5. The dying human heart. (14)
6. Various other toxic conditions effecting the myocardium.*
 - a. Influenza. (130)
 - b. Diphtheria. (130)

7. Rheumatic pericarditis and myocarditis. (125, 151) (see under section on RT changes for complete discussion of changes during the rheumatic process.) Porte and Pardee stress the "upward convexity" of the T wave, a factor which they say is not mentioned by

* J. B. Herriok commenting on Berman and Mason. (14)

Rothschild, Sacs, and Libman, Cohn, and Swift and others.

8. Pericardial effusion. (144, 7, 123) (see section on RT changes for complete discussion of this condition)

9. Certain drug poisonings.

- a. Digitalis. (38)
- b. Quinidine. (130)
- c. Emetine.

10. Hypertrophied athletic hearts. (130)

11. Normal hearts in various abnormal positions. (130)

While these conditions are quite numerous and would seem to be a basis for considerable confusion, they may usually be rather easily eliminated by careful history, physical examination, and laboratory work. Serial tracings will be of course of unquestioned benefit in arriving at a diagnosis.

C RS-T Deviations.

Probably the most characteristic feature of an acute coronary occlusion is the spectacular deviation from the isoelectric of the RT or ST segment. At first thought to be pathognomonic, it is now recognized that this abnormality may occur in various other situations. Among the most important of these are:

1. Rheumatic carditis - Cohn and Swift (31) observed cases of rheumatic fever in which there was an interruption of the down stroke of R and the beginning of a wave complex occupying the whole of the RT period. This abnormality persisted for several days and then returned to the normal. It was found present in both leads 1 and 3 of different subjects. Because this type of curve so closely resembled that of coronary occlusion, they concluded that the form

is not characteristic of the nature of the injury but is the result of any injury involving the heart muscle. They considered the contour of the curve an indication of the disturbance of certain functions of the muscle and perhaps dependent upon the site in the heart wall at which the injury takes place. Due to its transient character, they believe that the Aschoff bodies and ischemic areas consequent upon blood vessel involvement probably underlie the electrical disturbance to which the injured muscle gives rise. In many of their patients, they found prolonged isoelectric periods (PR interval), and in others upward, downward, or biphasic slopes of the RS-T segment. The alterations were, in many cases, "prolonged to affect the beginning of the T wave."

Parkinson and Bedford (122) also noticed coronary-like RS-T deviations and stated that they thought it was reasonable to suppose that these were due to a severe myocardial lesion. Sigler (152) observed frequent changes occurring in the tracings of cases of rheumatic myocarditis corresponding to the clearing up and involvement of new areas of heart muscle from time to time.

Despite the marked similarity of the tracings, rheumatic carditis may be easily excluded on the basis of the history and the age of the individual involved. In addition to this, it has been noted (Levine 95) that in rheumatic carditis the T does not arise from as high or as low a point on the R-S wave as is found in coronary thrombosis. Since this point is one only of degree, it is doubtful whether it will have much value in differential diagnosis.

2. Digitalis - The fact that this drug may produce alter-

ations in the RS-T segment has been cited by many investigators (122, 173, 20). Willcox and Loviband (173) state that the deviation produced may be in either direction, while Parkinson and Bedford (122) maintain that it is always negative, that it is never of opposite sign in leads 1 and 3, and that while the segment may be depressed, its origin is always isoelectric. If this is true, a typical "high takeoff" RS-T segment must always point toward the possibility of coronary disease even in the presence of a history of digitalis medication.

3. Hemopericardium - In the case cited by Scott et al. (144), the hemopericardium followed the rupture of an aortic aneurysm. There was no evidence of myocardial involvement at autopsy, and it was concluded that the changes were due to the increased pericardial pressure which, by decreasing the blood supply to the heart muscle caused anoxemia which was itself responsible for the changes in the electrocardiogram. In the case reported by Eliot and Evans (47), the etiology was the same, the tracing suggesting a type T3 of coronary record, with an elevated RT in leads 2 and 3.

4. Pneumonia. (47) (95)

5. Uremia. (47) (95)

6. Purulent Pericarditis - (144, 128, 7, 20) In the tracing of the case published by Purks (128), there was an elevated RT in leads 1 and 2 with an inverted T, "Which might well be classed as a coronary wave." This was accompanied by a moderate left axis deviation and a slurring of the QRS in lead 3. In discussing this case, the author stated, "It is true that with any case of pneumo-

coccio peritonitis there is an involvement of the myocardium for a depth of one or two millimeters, and thus we might explain the changes on this basis. It is also possible that partial annoxemia resulting from increased intrapericardial pressure played a part in producing the changes."

In 1934, Barnes (7) described the electrocardiographic changes which occur in patients in which actual occlusion is complicated by pericarditis. In the early stages, there is an elevation or upward rounding of the RS-T segments in all leads, which may be followed by inversion of the T waves. In some instances, it is followed by the development of a T pattern, "that can definitely be classified as a late relic of coronary occlusion." In the stage where the RS-T segment is elevated in all leads, he reported finding in some cases a typically developed Q pattern which he considered, "not only indicated infarction but also pointed to the location of the infarct." All of his tracings were of the "anterior" variety, the author being somewhat uncertain as to the possibility of posterior basal infarction of the left ventricle complicated by pericarditis producing the same picture. In concluding, he stated, "I am inclined to believe that the injury which the superficial layers of the myocardium sustain in the pericardial reaction accounts for the peculiar electrocardiographic changes, an interpretation supported by experimental evidence." (144, 10, 56)

7. Application of pitressin to the heart. (70)

8. Intravenous injections of pitressin. (70)

Gruber and Kountz conclude that in both types of pitressin

administration, the left posterior circumflex artery took part in the general vasoconstriction thus causing the change.

9. Stab Wounds of the Heart. (80, 12, 189) The reason for the form of the tracing in these cases may be attributed to the acute and serious injury of a large part of the ventricular wall (as in the record of Wilson et al, (189) or to the fact that branches of the coronary artery may be occluded by suture in the process of repair (as in the case of Bates and Talley (12). A return to normal in these tracings occurs sooner than in those of typical coronary occlusion with similar pictures, possibly because of the fact that they occurred in much younger individuals whose myocardium was in a relatively superior state prior to the accident.

10. Hypertension. (193, 168, 167) (For a complete discussion of the differential problems in hypertension see the end of this section.)

11. Congestive heart failure. (193)

12. Dissecting aneurysm of the aorta. (193, 103)
With rupture into pericardium or pleura. (115)

13. Hyper-parathyroidism. (193)

14. Changes in the blood sugar level found in

- a. Diabetic coma. (49)
- b. Insulin shock. (47)
- c. Abrupt but less serious changes in the blood sugar level associated with improper treatment. (157)

In diabetes the storage and/or utilization of glycogen is interfered with. Following excessive doses of insulin, the energy production of the heart muscle is handicapped because of the too

rapid lowering of the blood sugar and the depletion of the supply. In addition to the RS-T segment variations, there was noted (47) decreased amplitude and increased slurring of the QRS and a tendency for the ST and T wave to shift in a direction opposite to that of the major initial complex. Siegel (149) has shown that if the production of lactic acid from its precursors (sugar and glycogen) is interfered with (as in diabetes) the T wave becomes flattened.

15. Air embolism. (39)

16. Experimental Epinephrine injections - Douglas et al. noted that suitable doses of this drug can produce a marked displacement of the ST segment in the electrocardiogram of the cat. The deviation can be either above or below the isoelectric level. It can be so marked that the "monophasic" type of curve results. The changes produced by epinephrine can be abolished by nitroglycerine, and are probably the result of coronary spasm.

17. Rupture of the heart. (28)

18. Gumma of the heart. (129)

19. Non-penetrating thoracic trauma. (20)

20. Certain cases of congenital heart disease. (Tracings from the electrocardiographic department of the University of Nebraska College of Medicine.)

21. Pulmonary Infarction - The fact that the symptoms in this condition often resemble those of coronary occlusion makes similarity of the electrocardiographic tracings a most unfortunate situation. Love and Brugler (103) report several cases which closely resemble the T3 type of coronary infarction. McGinn and White

(108) and Barnes (9) differentiate the two conditions electrocardiographically as follows:

- a. S1 is rather constantly present in pulmonary infarction and apt to be wide, whereas it is usually absent in coronary thrombosis.
- b. ST2 is usually not elevated and commonly depressed in pulmonary infarction, and usually elevated and never depressed in the T3 type of infarction.
- c. T2 is low, diphasic, or slightly inverted in lung embolism, while it is definitely inverted in thrombosis.
- d. T3 is usually inverted in both conditions, therefore of little aid in the differential diagnosis.
- e. The precordial leads show a negative T wave in embolism of the lung whereas they are usually upright in the T3 type of coronary curve.
- f. Curves of pulmonary infarction show a tendency toward right axis deviation.

That many authors are not in accordance with the above stipulations is found in the fact that only 14% of the cases reported by Love and Brugler (103) and 33% of the cases cited by Soherf and Schonbrunner (143) conform. Love and Brugler believe that there is no electrocardiographic pattern typical of pulmonary infarction and furthermore, "That the electrocardiogram is not decisive at the present time in differentiating lung infarction from coronary thrombosis". A part of the factors responsible for the change in the electrocardiograph in pulmonary infarction arise from anoxemia of the heart muscle due to reflex changes in the coronary system, but one must not lose sight of the fact that the pre-existing state of the coronary arteries is also a factor to be taken into consideration.

Thus we see that acute changes in the RT and ST segments are usually associated with either

A. Sudden disturbances in the metabolism of the heart mediated through changes of certain constituent of the blood, or

B. With sudden partial or total deprivation of the blood supply to a portion of the myocardium. (47)

While the number of conditions mentioned above in which the acute changes of coronary occlusion may be simulated is rather large, relatively few will cause serious differential problems from a clinical standpoint.

D. Large Q Waves.

The following conditions have been cited as frequently exhibiting Q waves which correspond to the criteria of Pardee (121), Durant (43) and others.

1. Active rheumatic myocardial disease (121) due to a disproportionate damage to the left ventricular muscle.

2. Hypertension and cardiac arrhythmias (121). Usually show a complicating coronary arteriosclerosis affecting the myocardium of the left ventricle, with a large Q3 as the sole manifestation.

3. Normal Hearts - Accounted for on the basis of:

- a. Incorrect diagnosis. (121)
- b. Peculiar variations in the branching of the A-V bundle. (121)
- c. High diaphragm as in expiration, pregnancy etc. (53)
- d. Infants. (146)

4. Factors producing right axis deviation as

- a. Advanced rheumatic disease.
- b. Congenital pulmonic stenosis.

In these cases the relatively greater right ventricular hypertrophy causes a displacement of the septum and adjacent inferior portions upward and to the left, and the heart often rotates along its long axis so that the left ventricle lies more posteriorly with the right ventricle forming almost the entire cardiac surface. The rotation and displacement of the adjoining apical regions upward and to the left thus approximately corresponds to the direction of the early electrical axis calculated from the initial positive deflection in lead one and the negative deflection in lead three which always appear in this type of tracing. Hence the large Q3 may be due to considerable septal displacement, and therefore does not necessarily indicate any aberration in ventricular excitation such as occurs in myocardial infarction.

- 5. Severe hemorrhage from duodenal ulcer. (66)
- 6. Post-influenzal asthenia. (66)

Goldbloom and Kramer explain both of the above two cases on the basis that most acute infections cause temporary disturbance in the arterioles and capillaries, and that in this manner temporary myocardial dysfunction of sufficient degree to produce a deep Q3 wave might have been produced.

- 7. Hyperthyroidism. (66) (43)
- 8. Arteriosclerotic heart disease plus congestive failure. (43)

Thus we see that a large Q wave, the single most characteristic indication of an old healed myocardial infarction, may occur

in a variety of other conditions relatively easily differentiated on the basis of history and clinical examination.

E Increased Voltage of the T Wave.

Elevation of the T wave in one or more leads is commonly found in the following conditions.

1. Pneumonia. (18)
2. Rheumatic heart disease. (18)
3. Thyrotoxicosis. (78)

The type of elevation present here is usually easily differentiated from the characteristic upright "coronary T" wave, since it differs markedly in the contour of both the T wave and the ST segment. This variety has no rounded shoulders, its two limbs are not symmetrical, the descent being much steeper than the ascent, and the transition of its descent with the horizontal is much more abrupt than that of its rise. There is no "hump" in the ST segment. If these factors are kept in mind this variation should present little diagnostic difficulty.

F Ventricular Fibrillation.

In addition to sudden acute coronary closure, the following conditions are prone to result in fibrillation of the ventricles:

1. Early stage of chloroform anesthesia. (17)
2. Acute benzol poisoning. (17)
3. Electric shock with low voltage. (17)
4. Following intravenous adrenalin. (17)
5. As a termination in paroxysmal ventricular tachycardia. (17)

The general condition of the heart in these conditions differs

essentially from that in coronary thrombosis in that structural damage is not characteristically present. Because the termination of ventricular fibrillation is fatal, its appearance in electrocardiographic records is a rare occurrence.

G Precordial Lead Changes

Although to date they have been subject to less exhaustive studies, it has been recognized by many investigators that the coronary changes characteristically found in this derivation may be encountered in a variety of other conditions.

Absent R4 has been recorded in

1. Chronic glomerulonephritis. (107)
2. Acute glomerulonephritis. (107)
3. Rheumatic and syphilitic valvular disease. (107)
4. Pneumothorax. (107)
5. Thyrotoxicosis. (107)
6. Acute myocarditis. (107)
7. Bundle-branch block. (58) (197)
8. Circulatory failure as a result of thrombo-angitis of the smallest pulmonary vessels.

In the last condition anoxemia was probably the real cause.

In none of the above was there any evidence of myocardial infarction.

Negative T4 has been found in

1. Essential hypertension. (166) (See end of section for complete discussion of hypertension)
2. Chronic nephritis. (166)
3. Circulatory failure as a result of thrombo-angitis of the smallest pulmonary vessels. (65)

4. Healthy children. (166)
5. Digitalis. (173) In cases where a complete reversal does not take place a simple flattening may occur.
6. Heating or cooling the surface of the chest above the cardiac apex may cause inversion. (166)

Deviation of the RS-T segment has been described in.

1. Acute rheumatic myocarditis. (197)
2. Bundle-branch block. (197)
3. Digitalis - The dose required to produce the change is smaller in older than in younger persons. (197)
4. Circulatory failure as a result of thrombo-angitis of the smallest pulmonary vessels. (65)

A complete picture more or less resembling that found in the anterior type of infarction has been described in

1. Diabetes. (166)
2. Syphilis. (166)

A posterior infarction picture has been reportedly found present in a single case of

1. Rheumatic myocarditis.

Thus we see that the list of conditions producing coronary-like changes in the precordial leads is quite similar to that cited for the standard derivations. Here again, the history and clinical findings, when used in conjunction with the electrocardiogram, should make it possible to eliminate easily most of the interfering factors.

Probably the most confusing problem in the analysis of a tracing of suspected coronary disease is the proper evaluation of the changes which may be produced by hypertension. This becomes in-

creasingly difficult when one realizes the number of cases in which high blood pressure readings will occur in the arteriosclerotic type of coronary artery disease. In 1937, Strong and Caverhill (164) pointed out that the type of tracing described by Rykert and Roberts (138) is more likely due to myocardial damage, ischemia and fibrosis resulting from coronary sclerosis rather than being an expression of a strain produced by an elevated blood pressure.

The syndrome is as follows:

- a. Negative "coronary" T1. "Being shouldered and having a sharp apex angle".
- b. Reverse appearance of T3 - positive with increased amplitude.
- c. T2 may be positive, diphasic, or negative. When negative it resembles T1 in contour.
- d. QRS changes - RT segment is depressed in lead 1 and elevated in lead 3.
- e. Abnormal degrees of left axis deviation.

Bruenn (23) in commenting on the above also agreed that the T wave changes, especially when associated with arrhythmias and conduction defects were probably evidence of coronary sclerosis.

Van Nieuwehuizen and Hartog (167) further went on to show that there is often an absence of left axis deviation in hypertension which has brought about a heart dilatation and that this is due to factors which conceal the left axis deviation in these cases as

- a. Coronary effects (Pardee Q, M or W shaped ventricular complexes, "saddle-shaped" ST curves and, in the precordial leads, a tall R with a low S and a horizontal ST segment instead of a deviation from the isoelectric.
- b. Changes in the position of the heart.

- c. Notches distorting the ventricular complexes.
- d. By the tendency toward right axis deviation in mitral defects.
- e. Age - a deep Q wave being normal in youth.

They describe the picture of pure hypertension not due to aortic regurgitation, which will be considered later, as follows:

- a. Left axis deviation along, or.
- b. Left axis with a negative T1 and a positive T3 found usually in the cases with myocardial damage. or
- c. Left axis deviation with negative T1, positive T3, a convex ST1 interval and a concave ST3 interval. In such cases the heart usually shows pronounced dilatation.
- d. The absence of left axis deviation as a consequence of the factors mentioned above.
- e. Lead 4 - usually shows diphasic P, R shallow or absent, a normally wide ventricular complex, S rather deep, and T wave positive and of normal shape but usually of low voltage. There are usually no notches in the QRS complex.

The electrocardiogram of thrombosis of the left coronary artery followed by an anterior infarct which in lead 1 sometimes causes a convex, high takeoff of the RT segment with the T wave creeping into it (Smith, Pardee) changes in the course of time into a type in which the RT starts at the isoelectric level and continues after a rather long isoelectric course into a negative T wave which is comparatively narrow and shallow itself. (167) Such a tracing may resemble, at this stage, the hypertensive curve described above with the negative T wave in lead 1, but may be differentiated on the following points even in the presence of convex ST segments in both

cases.

- a. In hypertension, R after T1 has become negative is usually high (exceeding 12 mm.) while in the electrocardiogram of coronary thrombosis R1 is low.
- b. In the precordial leads the difference is marked. The typical picture of anterior infarction is usually characteristic enough to cause little difficulty in most cases, but it is possible that in certain cases of hypertension complicated by aortic regurgitation the electrocardiogram of the precordial leads may resemble that found in anterior infarction. In both cases, the R wave is small or absent and the S wave deep. In the coronary tracing, however, the ST segment usually has its origin far above the isoelectric line, and it is unusual to find a negative T wave of the type that usually accompanies anterior infarction in cases of aortic regurgitation. In addition, the ventricular complex of lead 4 in the infarction cases usually shows a notch (the size of which depends upon the location of the electrode) while this feature is frequently absent in regurgitation. The ascending ST segment is invariably straight in coronary thrombosis although may appear convex, as is usually the case in aortic regurgitation.

Because of the marked similarity of these two types of curves, however, the question arises especially in the case of aortic regurgitation, as to whether or not the coronary circulation has not been affected in addition due to syphilitic involvement of the intima.

In hypertension complicated by coronary sclerosis we find in the precordial leads a large R followed by a comparatively low voltage S wave, with an ST interval which is isoelectric in its first portion and followed by a T wave which is positive and of high voltage instead of the small R and deep S which are commonly present in the uncomplicated cases of hypertension. (167)

Thus it is evident that by detailed analysis the changes

due to hypertension may be recognized and evaluated even when they occur in conjunction with alterations due to involvement of the coronary circulation.

CASE REPORT

Since the method of taking precordial leads has only recently become standardized, it was thought that it might be of value to include a report of a recent case at the University Hospital in which a complete set of the multiple leads was taken according to recommendation (202, 203). In each case the electrode on the left leg was paired with an exploratory electrode* placed at the following locations:

CF1 - At the right margin of the sternum in the 4th interspace.

CF2 - At the left margin of the sternum in the 4th interspace.

CF3 - Midway between CF2 and CF4.

CF4 - At the point of intersection of the midclavicular line with the 5th interspace.

CF5 - At the level of CF4 in the anterior axillary line.

CF6 - At the level of CF4 in the mid axillary line.

In each case the galvanometer connections were made in such a way that the relative positivity of the precordial electrode is represented in the finished curve by an upward deflection and relatively negativity of this electrode by a downward deflection.**

* German Silver - One inch in diameter.

** To make the galvanometer connections in this manner it is necessary to connect the left-hand wire to the left leg electrode and the left-leg wire to the precordial electrode if the lead switch is set on lead 3. The only exception to this mode of application is in the taking of lead CF1 where the left leg wire must be connected to the left leg and the left-arm wire to the precordial electrode in order to obtain upright T and P waves as recommended by the standardizing committee. As the necessity for this reversal was not mentioned in the recommendations, I wish to emphasize here that the wire connections used in taking lead CF1 are reversed from those employed in obtaining the rest of the CF series.

The normals for these leads as found by the examination of a limited number of records in this department are as follows:

- CF1 - The P wave is usually upright but of low voltage. The QRS deflection is mainly upright, a small Q wave being followed by a large R. S is not present. The ST interval is normally isoelectric and followed by a T wave which is upright and of moderate voltage.
- CF2 - The P is of low voltage - may be positive, diphasic or negative. QRS is diphasic with an initial R wave followed by a relatively deep S. The ST takeoff may be slightly elevated normally, and the T wave which is strongly positive begins abruptly without the usual isoelectric period.
- CF3 - Essentially similar to lead CF2 except that R is relatively larger in proportion to S than in the preceding description.
- CF4 - As the exploratory electrode is moved to the left across the chest, there is an increasing tendency for R to become larger and S smaller, but considerable variation may occur without the tracing being considered abnormal. R must show a minimum deflection of 2 mm., and when the major component of the QRS deflection in this lead is upward, the minimum downward deflection allowable is 1 mm. It is never normal for QRS to be monophasic. The ST takeoff may be displaced upward a maximum distance of 2 mm. T is upright and of high voltage but rarely exceeding 10 mm. which is the upper limit of normal.
- CF5 - All the complexes are of relatively lower voltage than in the preceding leads. R usually exceeds S in height. The ST takeoff is commonly isoelectric but may be slightly elevated. T shows takeoff similar to above.
- CF6 - S is normally quite small. All deflections show relatively lower voltages than in the other leads.

Frequent notching is common in all the leads, M and W complexes occurring often. Normally no Q is present in leads CF2-6, but occasionally it may appear. When present, it is small and insignificant. T differs from its appearance in the standard leads,

taking off abruptly from the return stroke of S, being quite sharply peaked, and exhibiting relatively high voltage. P is small (never exceeding 2.5 mm.) Throughout and inclined to be quite variable although it is commonly positive.

History and Course of the Patient in the Hospital.

Mr. J. E., a white married laborer, age 69, entered the University Hospital for the second time 11/5 for the removal of a cataract. On 11/6 about 7.00 P. M., while lying quietly in bed, he was suddenly seized with a severe pain in the substernal region which he described as a "knife-like thrust" in his chest. This was followed by a "feeling of tightness" over the heart while the pain spread for a considerable distance down the inside of the left arm. He immediately became nauseated and vomited a small amount of bile. He felt very weak, appeared cyanotic, and had some difficulty in getting his breath. Examination at this time showed a pulse of 60 which was completely irregular. The heart sounds were reported as being too faint to be heard, and no friction rub was reported. The blood pressure dropped from 135/80 to 100/70 during the attack. The patient was immediately given morphine grs. $\frac{1}{4}$ following which he slept. On awakening the next morning, he still had soreness in the substernal region which persisted in varying degree for some days. Immediately following the attack, the patient's temperature was recorded as 97, but it rose to 100.4 on 11/8 and remained at 100.0 on 11/9. The pulse rate rose steadily, reaching a maximum of 90 on 11/9 and then gradually dropping back to normal. A blood count taken 11/14 showed 10,700 white blood cells. On 11/18, the

leucocyte count was approximately the same (11,100). A sedimentation rate run on 11/16 revealed that one centimeter of blood settled twenty-four millimeters in one hour and forty-five minutes and by 11/18 this time was increased to two hours and ten minutes. After several days, the patient felt so well that it was difficult for him to remain in bed. On 12/5, a discission of the right eye was performed followed on 12/8 by extraction of a left cataract. His convalescence was uneventful, and he was dismissed from the hospital on 12/22/38.

EXPLANATION OF SERIAL ELECTROCARDIOGRAMS.

The first electrocardiographic record was taken on 11/8 approximately thirty-six hours following the accident. The remaining records were obtained at frequent intervals as shown by the figure. The auricular fibrillation appearing in the first tracing was replaced on 11/9 by a regular sinus rhythm. The graphs of 11/12 and 12/5 show numerous extra-systoles of left ventricular origin. The consecutive changes in the PR interval were as follows:

11/8---auricular fibrillation.
11/9---.20 sec.
11/12---.21 sec.
11/15---.22 sec.
11/25---.22 sec.
12/5---.20 sec.

Unfortunately it was not possible to have this man report back for check up tracings after he left the hospital. Below is an analysis of the serial records according to leads.

Lead 1. On the 11/8 a typical monophasic complex was evident. The ST segment showed less elevation on 11/9 and had reached

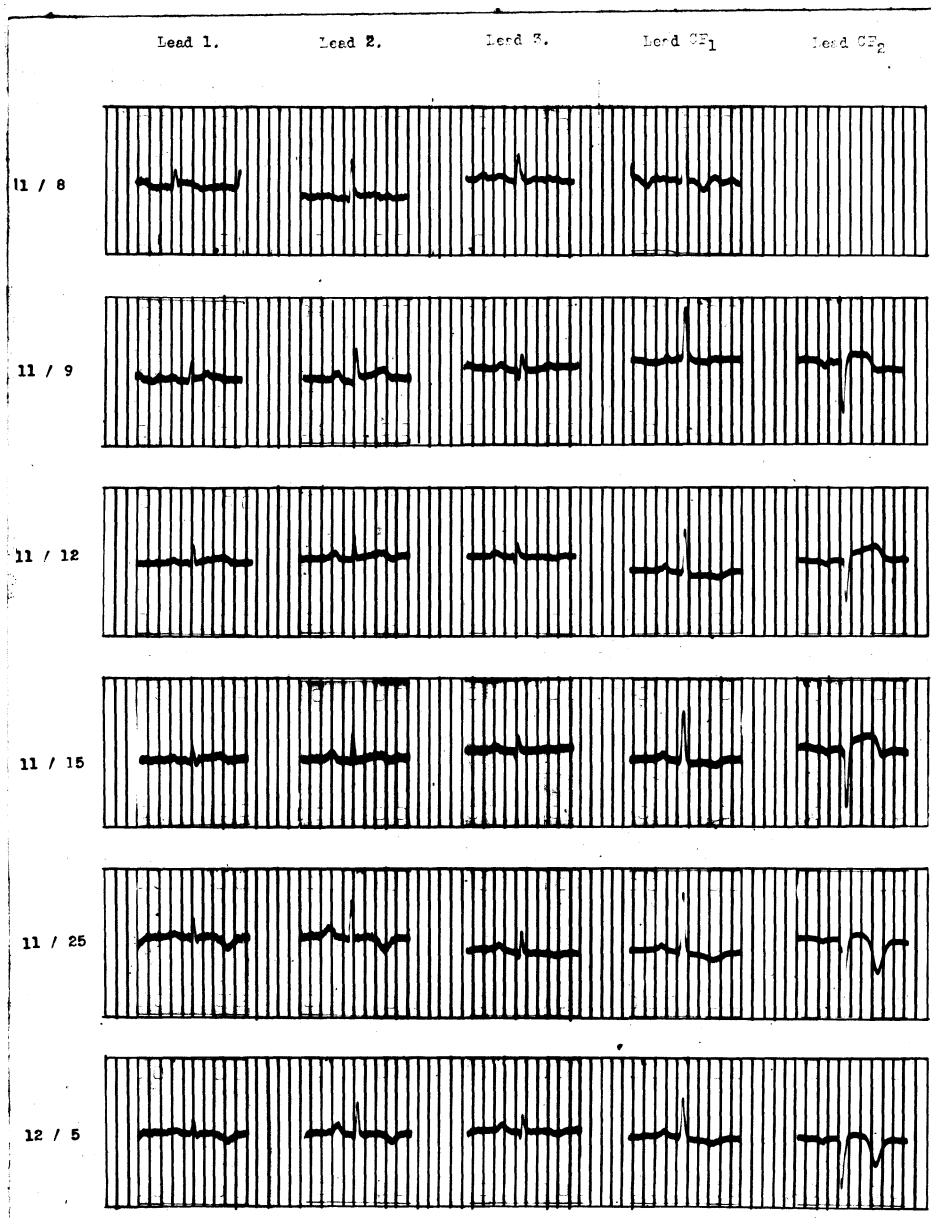


Figure 4.

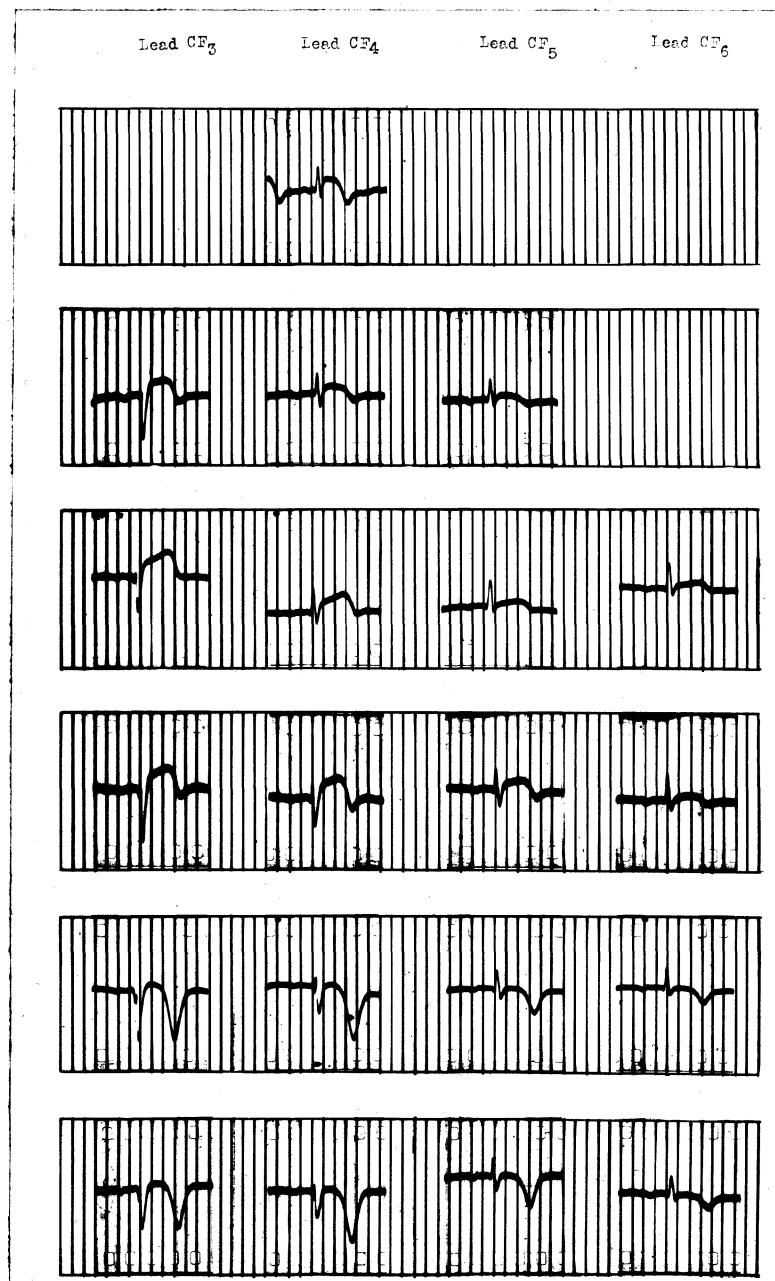


Figure 4 (Cont'd)

the isoelectric level on 11/12. By 11/15, the starting point for the subsequent T inversion was definitely visible at the end of the ST hump, and by 11/25, negative T1 was well developed. On 11/12 and again on 11/25, there is a suggestion of an initial downward deflection but no definite* Q1 appeared at any time. The deflections of this lead show relatively low voltage throughout.

Lead 2. The ST elevation in this lead is most marked on 11/9 and by 11/12 has reached the isoelectric level. Beginning T inversion is noticeable on 11/12 and by 11/25, the typical coronary T3 is fully developed. The P waves show progressive enlargement reaching a maximum on 11/25. There is a suggestion of a negative initial ventricular deflection on 11/9 and again on 11/25 (compare with lead 2). The tracing with lowest voltage is recorded on 11/12.

Lead 3. The ST segment changes in this lead are negligible. A Q wave is present on 11/9 and also on 11/12 which is 25% of the height of the largest R (R of lead 2) for this date. The low voltage positive T wave on 11/9 progresses through the isoelectric to a negative deflection by 12/5. The lowest voltage recorded for this lead was on 11/12.

Although a diagnosis of anterior infarction must be made after an examination of leads 1 and 2, the changes which might be expected in this type of occlusion are not present in lead 3. Instead we have the development of a large Q3 and the tendency toward the development of a negative T deflection, both characteristic of

* According to the criteria of Durant (43)

posterior involvement. All axis readings for the standard leads were within the normal range.

Lead CF1. The QRS is mainly monophasic and upright, but the suggestion of an initial Q persists throughout. The T wave, which is sharply inverted on 11/8, returns to the isoelectric line on 11/9 and then shows a tendency toward delayed inversion in subsequent records. On all dates but 11/8, there is a definite slurring present near the upstroke of R. The lowest voltages recorded are on 11/12 and 12/5.

Lead CF2. The P waves are inverted throughout. The QRS is monophasic and downward in all records. The ST segments are elevated with the formation of monophasic complexes on 11/9 and 11/12. The beginning of the development of a negative T is noticeable on 11/15 with a full-blown, high voltage, inverted coronary T appearing on 11/25. There is a definite notching of the beginning of the downstroke of Q in all records. Lowest voltage is recorded in 11/12.

Lead CF3. The changes are practically identical to those described for Lead CF2. It is interesting to note the small negative deflection, appearing at the point where the elevated ST segment returns to the isoelectric line, which is first noticeable on 11/9. There is some suggestion of the same type of wave occurring in Lead 2 for the same date. In both cases it disappears on 11/12 only to appear again on 11/15 and develop into a definite coronary T by 11/25. The inverted, sharply peaked, T waves for both 11/25 and 12/5 exceed two millimeters in depth and thus must be classi-

fied as large T4's. The lowest voltage is recorded on 11/12.

Lead CF4. The P waves are of low voltage and diphasic in all leads. The elevated ST segment shows the beginning of a negative T wave at the return to the base line in 11/8. Both the deviation and the negative deflection are less evident on 11/9, and on 11/12, the elevated ST rises to a peak before returning to the base line. On 11/15, the deviation is again more marked and the beginning T wave has reappeared. By 11/25, the ST segment has returned to the isoelectric and a large, inverted, sharply peaked T4 is present. The accompanying changes in the QRS complex are quite interesting. On 11/8 a large R is followed by a relatively small S. On 11/9, the S has increased in size while the R has decreased: and by 11/12, the suggestion of a small Q wave is present. The last tracing in the series for this lead (12/15) shows a Q followed by a very small R, the latter corresponding to the notch on the downstroke of Q present in Lead CF3 for this date. The lowest voltage was recorded on 11/9.

Lead CF5. The changes here are similar to those in lead CF4, but are less marked. R remains dominant to S on all dates, the largest deflection of the latter appearing on 11/15. An inverted T appears on 11/15 and reaches full development on 12/5 but does not exceed 10 millimeters at any time. Lowest voltage is recorded on 11/9 and 11/25.

Lead CF6. The changes here again resemble those appearing in Leads CF4 and CF5 but are even less marked. R is relatively large throughout and S reaches a depth of two millimeters on only one oc-

casion (11/15). A small negative T wave is present on 11/25 and again on 12/5. Lowest voltages are recorded on 11/25 and 12/5.

From examination of the chest leads, it is evident that Lead CF4 is probably the most advantageous one for study since, in this case at least, it shows the most marked alterations, although Lead CF3 is almost if not equally as good. From the analysis of the precordial leads, a diagnosis of anterior infarction must be made.

The value of the precordial leads is well illustrated when one considers that no significant abnormalities are present in the standard leads taken on 11/12 and 11/15. These records were taken during the period of transition between the ST segment and T changes, when the elevation of ST had again reached the isoelectric level but before definite final ventricular deflection changes were manifest. The chest leads on these dates, however, showed well developed, significant abnormalities characteristic of anterior infarction.

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